circulatory disturbance. This was manifested by a marked fall in both the maximum blood-pressure and in the pulse-pressure.

In the less strenuous type of exertion we usually find an increased urinary acidity, albuminuria, cylindruria, and at times blood cells.

A critical analysis of these findings, however, shows, first of all, that in the more strenuous effort the amount of albumin, the presence of blood cells and the number and type of casts all depict a more serious renal disturbance than is found in the less strenuous form of exercise.

Urinary acidity was increased after exercise in 85 per cent. of the cases, but it did not occur more frequently, nor was the acidity higher in the severe exertion cases than in the milder ones.

There is a positive relationship between the degree of albuminuria and cylindruria and the type of physical exertion; but these observations show that no such relationship can be established between the urinary acidity and the occurrence of albumin, casts and blood cells in the urine.

THE ASSOCIATION OF FEVER WITH FRACTURE OF THE SKULL.

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A slight rise of temperature is so common after fracture of the skull—with and without injury of the intracranial contents—that one rather expects it in almost every case; the rise is rarely above 100° F., is short-lived and lasts for approximately twenty-four hours, after which subsidence to the normal occurs and the latter continues until convalescence is completed. In some of the patients, however, fever assumes a more important role: it rises to extraordinary heights; it persists for comparatively long periods of time; it indicates some grave complication or the reaction to an extensive trauma. The cases in this latter group form the basis for this communication.

Such fever occurred in 15 of a series of 72 cases of fracture of the skull which were recently studied—21 per cent. In another series of cases of fracture of the skull, admitted to the hospital in a different period of time, 22 patients of a total of 77 developed fever—29 per cent. In the 15 febrile cases of the first series the fractures were situated in the posterior fossa in 6, and in the middle fossa in 2; in the others the fractures were distributed over the vertex and sides of the skull. One fracture was compound externally; one communicated with the middle ear and 2 with the nasal cavities; the others were all closed fractures. Eight of the patients who developed fever died; in 4 of these the cause of death was a meningitis.

The cases which developed fever can be divided into the following groups:

1. The Ordinary Cases:

In a number of the febrile cases the temperature was of a moderate degree and the cause for the latter could not be established. In one of the cases (No. 192204) fever was present for two days. Except for the fact that it persisted somewhat longer than usual it approximated to the initial reaction so frequently seen and would, ordinarily, draw no extra comment. In another patient (No. 156919) there was an initial rise of temperature which fell promptly as it usually does; on the fourth day it rose again to a moderate height to fall to the normal after twenty-four hours more. Both of these patients recovered and the cause of the temperature remains obscure.

Case No. 192204. A boy, aged seven years, fell from a fire-escape a distance of thirty feet and landed on the front and side of his head. For a period of thirty minutes he was unconscious; thereafter he vomited a number of times and complained of pain in the left side of his head. The physical examination showed a hematoma of the scalp and the roentgen ray showed a frontoparietal fracture. The neurological status was normal except for some hyperactivity of the knee-jerks. The character of the fever is indicated in Fig. 1. The patient made an uninterrupted recovery.

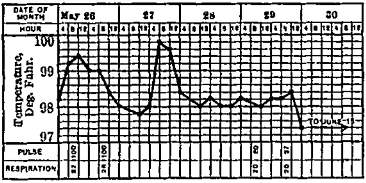


FIG. 1

Case No. 156919. A boy, aged seven years, fell a distance of eight feet and struck the right side of his head. He was unduly quiet thereafter and complained of a dull headache; he vomited after taking some food. There was a hematoma of the right parietal region and the roentgen ray demonstrated that the fracture lay directly underneath and extended into the mastoid. There were no neurological disturbances. The fever is shown in Fig. 2. An uninterrupted recovery followed.

2. The Fatal Cases:

In the following case an excessive degree of fever was associated with an extensive cranial fracture and with widespread disorganization of the intracranial contents. The notes of the case follow.

Case No. 179660. A six-year-old girl who was thrown from an automobile struck the pavement with the top and back of her head. There were several large hematomata over the occiput and vertex and behind the ear. The neurological disturbances which

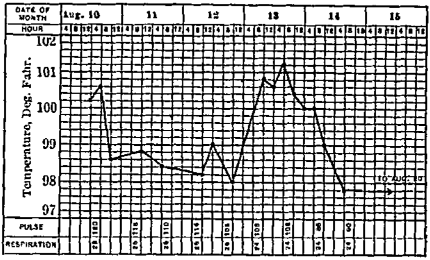


Fig. 2

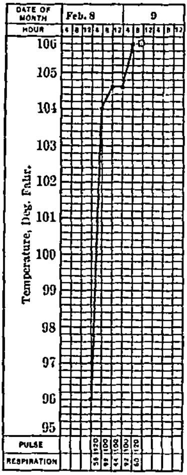


Fig. 3

pointed to a brain injury included (1) unequally dilated pupils, neither of which reacted; (2) internal rectus paralysis; (3) beginning papilledema; (4) spasticity of all four extremities, with absent abdominal and exaggerated limb reflexes and elicitable abnormal reflexes, including Babinski, Gordon and Oppenheim responses; (5) peculiar athetoid movements of the hands, with flexion of the hands at the wrists; (6) stupor, with Cheyne-Stokes breathing. At the time of death—about twenty hours after the injury—there was (7) complete flaccidity, with (8) absent reflexes and (9) widely dilated pupils. Prior to death the temperature reached 106.8° F. (Fig. 3).

The cause of death in this child was evidently a widespread disorganization of brain substance, aided by a marked grade of intracranial compression from edema and hemorrhage. The temperature, owing to the shortness of time elapsing between the injury and death, assumes the characteristics of an antemortem rise. Our knowledge in this regard is, however, very deficient, and it is quite probable that this as well as other antemortem rises of temperature have something in common with disturbances of the heat-regulating centers.

The notes of two other fatal cases are given in which fever formed one of the prominent parts of the symptom-complex.

Case No. 190666. A fifty-five-year-old patient sustained a bad crushing fracture of the right malar region and side of the skull immediately opposed to it; there was very marked protrusion of the orbital contents. Besides the last the clinical picture included much shock; marked restlessness; fever (Fig. 4); a sluggish, irritable and disturbed mentality, which changed to stupor, deepened to coma, and terminated in death forty-eight hours after the injury was sustained. The character of the symptoms, taken in conjunction with the location of the fracture, pointed to disturbances in the frontal lobe.

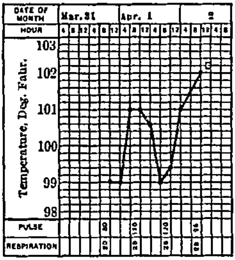


FIG. 4

Case No. 173080. In the second of these two patients, a woman, aged sixty years, a fracture of the base of the skull was associated, clinically, with rigidity of the neck, coma, general spasticity and loss of control of the sphincters. Death occurred seventy-two hours after the injury. Fever (Fig. 5) was present during the entire period the patient was in the hospital.

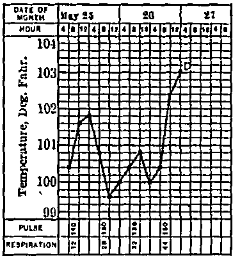


FIG. 5

In both of these patients the probabilities are very strong that there was considerable intracranial hemorrhage; in the second patient the symptoms indicated cerebral compression; in neither of them was there any indication present of any infectious process or of any complicating condition which could account for the irregular temperature.

3. Fever in Compound Fractures:

In one patient fever, as noted in Fig. 6 (Case No. 185032), was associated with a compound fracture. The scalp wound had been

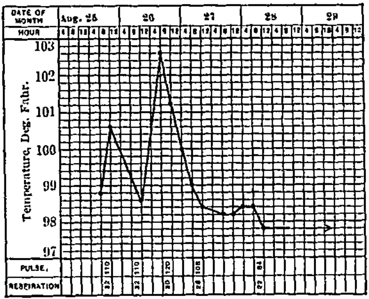


FIG. 6

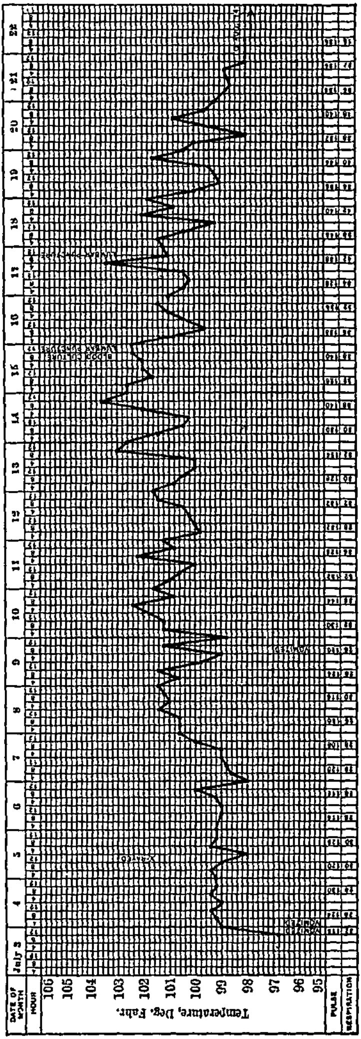
sutured immediately and, apparently, had healed by primary intention. In the absence of any other cause it is quite likely, however, that infection was present in the wound and was combated by the natural powers of the body without any disturbance of the healing.

A very remarkable case is the following:

Case No. 193199. The patient—a child, aged six years—sustained a fracture in the posterior fossa of the skull. Nothing extraordinary was noted at the time of admission to the hospital, and there were no signs of disturbed neurological function. Fever of a slight grade (Fig. 7) was present from the beginning, and by the fifth day it had risen to 102.4° F. During the course of the routine examination a laceration was discovered in one drum-head, and owing to the positive history of no preceding affection of the ear the otologist (Dr. Braun) rendered the opinion that the fracture had opened into the middle ear and, presumably, had become secondarily infected. The otitis subsided very quickly, however, and ten days after the injury the perforation in the drum-head had closed completely. On the thirteenth day the temperature reached 104° F. Lumbar puncture had hitherto not been practised, owing to the recent work of Weed and his co-workers, and of Wegeforth and Latham, but at this point it was done, and was subsequently repeated two days later; except for some increase of pressure on the first tapping the procedure yielded negative information both times: there were 3 cells per cubic millimeter and the fluid was bacteriologically sterile.

During the second week the following neurological signs developed rather slowly and irregularly: (1) A weakness of one facial nerve; (2) a convergent squint; (3) slight twitching of the left upper extremity; (4) very slight retraction of the head, with moderate rigidity of the neck which could, however, be forcibly overcome; (5) tendency to a Kernig on the right side; (6) irritability. No abnormality could be discovered in the chest or abdomen and no focus of infection could be demonstrated in any of the limbs. In the third week the temperature gradually subsided to normal, the signs and symptoms all gradually disappeared and thereafter the child made a perfect recovery.

The question in this particular case was whether the child had had a basilar meningitis, from which it had recovered, or whether the fever was due to some other cause. The probabilities of a meningitis were much strengthened by the fact that the patient was admitted into the hospital a short time after the termination of the influenza epidemic; coincident with the stay of this patient in the hospital another child had been admitted to the same ward and had gone through a similar clinical picture—in which similar negative laboratory findings were demonstrated—and, after it had died, an autopsy revealed a basilar meningitis, with a shaggy, greenish exudate in which influenza-like organisms were demonstrable in smears. The physical findings were in no way conclusive, inasmuch as cases of basilar or posterior] fossa fractures quite


DATE OF MONTH
HOUR
Temperature, Deg. Fahr.
PULSE
RESPIRATION
July 3
4
5
6
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12
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17
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22
106
105
104
103
102
101
100
99
98
97
96
95
VOMITED
VOMITED
X-RAYED
VOMITED
BLOOD CULTURE
LUMBAR PUNCTURE
LUMBAR PUNCTURE
DIED

FIG. 7

commonly exhibit some of the signs of meningeal irritation. The probabilities, when all things are considered, seem to be that the physical findings were due to irritation of the meninges by the extravasation of blood and that the fever was due to absorption of the latter and to the aseptic inflammatory processes necessary to the proper repair of the fracture.

4. Fever Due to Complications:

Fever occurred also as a reflection of some complicating condition. The most prominent of these was a meningitis. The two cases given as examples show the varieties of fever which were present when this complication appeared after fracture of the skull; in the one (No. 177900) the infection was fulminant, the fever was high and the clinical course was very short and terminated in a fatality; in the other (No. 173371) the symptoms and fever made their appearance after several days and the course was more protracted.

Case No. 177900. The patient had a fracture in the frontal region and the roentgen ray showed, clearly, the communication with the frontonasal sinuses. The signs of meningitis were developed fully within twenty-four hours and the spinal fluid was loaded with polymorphonuclear cells and contained type I pneumococci. Fig. 8 shows the character of the fever.

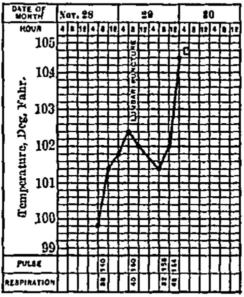

FIG. 8

Case No. 173371. In falling a five-year-old lad struck the pavement with the back of his head. He vomited and became unconscious; later this disappeared. The eyeballs showed some nystagmus when turned to the right, otherwise the neurological status was normal. Three days later the temperature rose to 105° F. The patient became apathetic; he assumed an attitude of complete flexion; there was moderate opisthotonos, a bilateral Kernig, and a suggestion of a Babinski. The spinal fluid contained many cells and Type IV pneumococci. The patient died.

The postmortem examination showed a fracture extending to the left jugular bulb and into the left middle ear and a general purulent meningitis.

Fig. 9 shows the temperature curve.

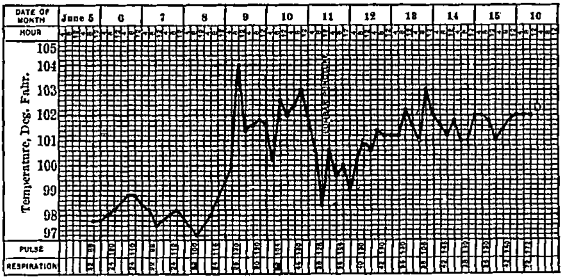

FIG. 9

5. Fever in the Operated Cases:

Fever in the operated cases was present in some both before and after operation and in others only after operation. In the latter it was frequently so because the patients were operated upon immediately after admission to the hospital and there was no opportunity allowed before the interference for the temperature to rise. When excessive fever developed after operation there was never any conclusive proof that it was directly due to the operative intervention, and, in all probability, the nature of the injury and of the resultant pathological changes contributed largely, if not entirely, to the pyrexia. The following cases are good examples of those in this group.

Case No. 161336. One hour before admission the patient fell down an elevator shaft. The patient was pale, semiconscious and in marked shock. In the parietotemporoöccipital region was a large hematoma. Blood was escaping from the ear. There was a progressive lowering of the pulse-rate to forty-four beats to the minute. The neurological status included (1) an initial period in which the reflexes were absent; (2) periods of excitation and stupor alternating with one another; (3) paralysis of left arm and leg; (4) reappearance of the reflexes which became hyperactive; (5) clonus at both ankles.

The operation, which was done four hours after the injury, showed a fracture extending from the occiput into the temporal bone and mastoid and which was depressed in areas. The dura was plum-colored and there was a perforation in the lateral sinus. An extensive subdural hemorrhage was also present and the brain was badly lacerated. Bilateral subtemporal decompression was done.

On the following day improvement was present in that power began to return in the arm. Twenty-four hours later, however, the patient's condition became worse and death followed shortly. The character of the fever is shown in Fig. 10.

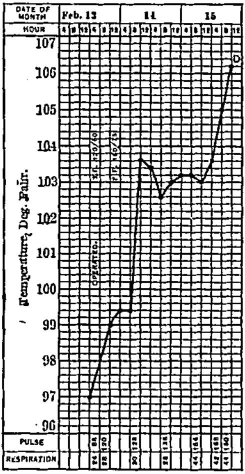

FIG. 10

Case No. 148405. A boy, aged nine years, was run over on the street and was brought immediately thereafter to the hospital. On admission the boy was in stupor and was bleeding from the nose and mouth. The neurological findings included (1) left facial weakness; (2) twitchings of left upper limb; (3) convulsions beginning in the left arm, spreading to the left leg and to the opposite side of the body. Within the next twenty-four hours the signs of compression grew more marked.

Operation[1] was done on the day following the injury. A flap operation was done. The exploration showed (1) that no hemorrhage was present; (2) great tension and edema of the brain; (3) some bloody serum in the basilar portion. No improvement followed the operation and the patient died approximately twenty-four hours later.

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[1] The reasons for the operation and for the exploration through a bone flap are not apparent from the history.

In Case No. 148405 fever (Fig. 11) was present from the time of admission to the hospital and reached 102.8° F. within twelve hours. At the time of operation—forty-eight hours after the injury—the temperature had reached 106.6° F. There was marked hyperpyrexia at the time of death. The injury included a basilar fracture and much disorganization of brain tissue, as evidenced by the great tension and widespread edema of the cortex.

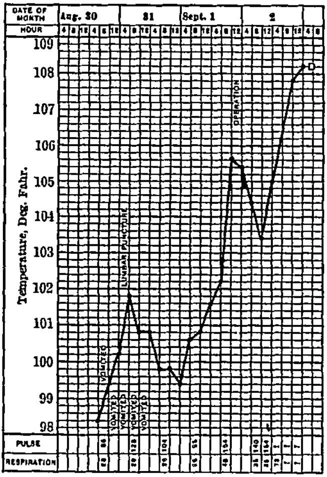

FIG. 11

Cases of operative infection are, of course, not included in this discussion.

6. Fever after Lumbar Puncture:

In one of the patients fever occurred after lumbar puncture. A child sustained a fracture in the parietal region. On admission the patient was drowsy but otherwise exhibited no definite symptoms of any focal lesion. Twenty-four hours later there were indefinite signs referred to the lower extremities: the left seemed to have impaired power, and the right was somewhat spastic. Lumbar puncture was done at this point and some bloody fluid was released from the spinal canal. Up to this time the temperature had been normal, but immediately thereafter it began to rise and in an additional twenty-four hours it reached 101.4° F. Operation, which was done at this time, demonstrated extradural bleeding over an intact dura, and was followed, shortly, by a fatality (Fig. 12).

COMMENT. Fever associated with injury of the skull and of the intracranial contents occurs much more commonly than a perusal of the literature would lead one to believe; and while in the presence of some definite infection or other lesion the origin of the temperature is perfectly apparent, in others the cause for the fever is obscure and the mechanism for its appearance, especially when hyperpyrexia is present, is ordinarily said to involve disturbances of the heat-regulating centers.

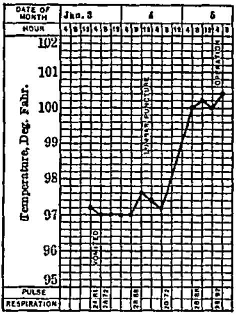

FIG. 12

It was most instructive to compare with these cranial and intracranial injuries a series of cases in which a fracture of the skull could be ruled out but in which the character of the symptoms and the physical findings indicated that a state of concussion (brain contusion) was present. It was cause for much surprise to learn that between 20 and 25 per cent. of the latter cases developed fever of one kind or another during their stay in the hospital. As a general rule the fever was of a very moderate kind and compared with the variety described in group 1. In a very small minority the temperature rose to greater heights. The following cases form excellent illustrations.

Case No. 181734. A young child was thrown in an automobile accident and was brought immediately to the hospital. Except for a small hematoma in the scalp nothing abnormal could be detected and within twenty-four hours the child was perfectly normal, with the exception of the low grade of fever noted in Fig. 13. No adequate cause for the temperature besides the trauma to the head could be discovered. The diagnosis was "concussion."

Case No. 186469. A man, aged forty-three years, was struck in the head by a moving car forty-eight hours before admission to the hospital. He has been in a very dazed condition since, has complained of much headache and has vomited a number of times. A complete examination in the hospital showed a normal neurological status. With rest in bed the symptoms rapidly disappeared and forty-eight hours later when the man left the hospital no abnormality of any kind could be distinguished. There was absolutely no evidence of any skull fracture.

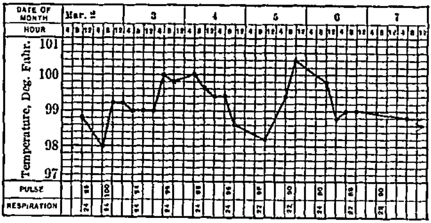

FIG. 13

The fever which this man developed in the hospital is extraordinary. Immediately on admission the temperature (Fig. 14) was 102° F.; within a few hours it rose to 104° F., and thereafter it gradually fell until the time of discharge from the hospital. No other cause beside the head trauma could be demonstrated which could adequately account for the fever. The final diagnosis was "concussion."

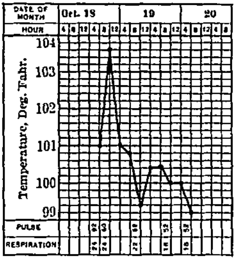

FIG. 14

When fever appears in the cases of this kind, as well as in those in which the brain contusion is associated with fracture of the skull, the assumption seems justified that the more or less widespread disorganization of brain tissue has, probably, primarily involved the heat-regulating centers, and that any fever which appears is due to the latter disturbance. Implication of these centers could very possibly result secondarily from a general intracranial compression resulting from a sudden cerebral edema following the trauma or more slowly when the compression is due to other causes. Fever under these circumstances would be liable to be a hyperpyrexia. On the other hand there are cases with a more moderate or low grade of fever in which the symptoms and physical findings indicate a more restricted disorganization of brain tissue; possibly in these the temperature finds its origin in the inflammatory process (encephalitis?) essential to the proper repair of the injury.

It is quite possible that in some of the patients with fracture of the skull the phenomenon of fever is connected with the absorption of extravasated blood. In fractures of the long bones an extensive hematoma formation not infrequently is accompanied with a low grade of fever which persists for a short time.

In a series of 61 uncomplicated and closed fractures of the long bones fever was present in approximately one-third. The fever was of a very low grade, usually varied between 100° and 101° F., very rarely reached above 101° F., and never presented the bizarre forms described above with the cases of fracture of the skull or concussion of the brain.

Confirmation of this observation is found in the clinical courses of hematomata of the buttocks: quite commonly these are associated with an intermittent fever which is liable to continue to mount to 103° and 104° F. daily in the absence of any demonstrable infectious or suppurative process until the clot is totally absorbed. A drop of the temperature frequently follows the aspiration of the liquefied portions of the clot, and it is often necessary to incise freely into the hematoma before the temperature disappears; evidently the fluid carries with it some factor which serves to keep up the fever. It seems quite reasonable to assume that a similar mechanism is functionating in some of the cases of fever following a head injury, and the assumption seems especially applicable to the cases of basilar fracture, with hemorrhage into the middle and posterior fossæ of the skull. In these patients lumbar puncture almost invariably releases a bloody fluid which is bacteriologically negative, and which, microscopically, shows very few cellular elements outside of the red blood cells; and the indicated findings serve to negative any impression of an infectious meningitis which might seem probable because of the exhibition of more or less marked symptoms of meningeal irritation. As anticipated in the notes of Case No. 193199—which seems to be an excellent example of this combination of circumstances—the probabilities are very strong that the fever owes its origin to the absorption of extravasated blood, and the physical findings indicate irritation of the meninges by the presence of a foreign fluid.

Our attention to the question of fever after cranial and intracranial injuries was stimulated by some work of Weed and his co-workers. Briefly stated their work consisted of the production of an artificial bacteremia, after which cerebrospinal fluid was released by lumbar puncture; the procedure was followed by a cerebrospinal meningitis. Confirmation of this experimental work in clinical medicine was recently reported by Wegeforth and Latham, who found in a number of general and pulmonary pneumococcus infections that the release of normal cerebrospinal fluid by lumbar puncture was followed by meningeal infection; they describe five cases of their own and refer to others in the literature. Our Case No. 188349 seems to belong somewhere in this group; in this patient a lumbar puncture, with the release of some bloody fluid, practised approximately twenty-four hours after a cranial and intracranial injury, was followed by a rise of temperature. Apparently this seems to be a mild case of infection which, perhaps, under another subsequent course of events might have spontaneously disappeared. The source from which the infection took its origin could very possibly be a dormant one and lie hidden in the tonsils, the teeth or other similar locality. The absence of any true purulent meningitis would seem to indicate that mild cases of infection can occur from which retrogression can be spontaneous. Since the appearance of the communication of Weed we have been very careful and very backward about doing lumbar puncture after cranial and intracranial injury.

In considering the mechanism of fracture of the skull, with intracranial injury, attention was attracted to the fact that a release of cerebrospinal fluid must also occur into the adjacent tissues when a communication is established by dural laceration with the subarachnoid space. An analogous situation is established to the release of cerebrospinal fluid by lumbar puncture; and it is not unreasonable to assume, further, that in the presence of some hidden or dormant focus of infection in the body, either nearby or at a distance, fever would follow. The assumption offers a very satisfactory explanation for the initial rises of temperature which are commonly found after fractures of the skull; it offers an equally plausible explanation for the sudden and sharp rises of temperature which are sometimes seen to follow decompression or other cranial operations in which the dura is opened and cerebrospinal fluid is released from the subarachnoid space. The fact that the process does not terminate always in a suppurative stage should not appear extraordinary, and the retrogression is probably due to the fact that the organism is possibly accustomed to the variety of infection which is lighted up or that the body's natural resistance powers are amply sufficient to quickly bring the process under control. In any case the insult of lumbar puncture, trauma or operative interference, with a subsequent release of cerebrospinal fluid, seems to create a *locus minoris resistentiæ* and the injured area forms a fixation-point for any pathogenic organisms which, at the moment, are circulating in the blood-stream.

It seems very probable, when all things are considered, that in any particular case more than one of the factors indicated in this communication may be operative to account for any fever which develops. Only in a comparatively few cases is it possible for one to put one's finger accurately upon the actual mechanism.

SUBCUTANEOUS EMPHYSEMA: A COMPLICATION OF INFLUENZA PNEUMONIA. REPORT OF SEVEN CASES.

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CHICAGO,
AND
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(From the Cantonment Laboratory, Base Hospital, Camp Zachary Taylor, Ky.)

Recent clinical reports have mentioned subcutaneous emphysema as a rare complication of influenzal bronchopneumonia. Thus, Symmers[1] records 3 cases; Berkley and Coffen[2] 11; Clark and Synnott[3] 12; Blanton and Irons[4] and Stone and Swift[5] merely mention its occurrence. Leichtenstern,[6] in his exhaustive monograph on influenza, makes no note of this complication. Our own experience comprises a total of 9 cases, of which we have studied 7 cases carefully, and these are herewith presented. In about 3000 cases of influenzal pneumonia subcutaneous emphysema occurred in 0.3 per cent., and in the entire series of over 12,000 cases of influenza in only 0.07 per cent.

Clinical Features of Influenza Pneumonia. The clinical picture of influenza pneumonia has been so often described that we do not deem it necessary to repeat it here. Our experiences, on the whole, correspond to all previous reports. We desire to emphasize, however, certain features of the disease, not only because they were apparently characteristic of the picture but because they may be significant factors in the production of emphysema.

1. *Toxemia.* This was striking early in the epidemic and entirely out of proportion to all physical findings.

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[1] Jour. Am. Med. Assn., 1918, lxxi, 1482.
[2] Ibid., 1919, lxxii, 535.
[3] Am. Jour. Med. Sc., 1919, clvii, 219.
[4] Jour. Am. Med. Assn., 1918, lxxi, 1988.
[5] Ibid., 1919, lxxii, 487.
[6] Influenza, 1912. Alfred Hölder, Wien u. Leipzig.